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Inhalation of Cold Air.—Dogs were caused to breathe extremely cold air for periods ranging between twenty and one hundred and thirty-three minutes. The cold air was brought into the mouth of the animals through a vacuum-jacketed (Dewar) cannula. The temperature of the air was between −50 and −28°C. when it reached the larynx. When the air reached the bifurcation of the trachea it had been warmed to a temperature of above +18°C.; and when it left the lungs it was within 1 or 2 degrees of normal. The rectal temperature dropped in no animal. The only ill effects were hyperactivity of the mucous glands of the upper respiratory tract, coughing and hoarseness. One animal died of obstructive edema of the pharynx and larynx, caused by direct contact of the cold tip of the laryngeal cannula with the adjacent tissues. In the animals which were killed between four and twelve hours after the exposure there was some edema of the mucous membrane of the larynx, excessive secretion of mucus throughout larynx and trachea, small plate-like zones of atelectasis and subpleural zones of emphysema throughout both lungs. In the animals killed twenty-four hours after exposure there was damage of laryngeal and tracheal epithelium, small plugs of mucus mixed with desquamated epithelial cells in some of the smaller bronchi, plate-like atelectasis and emphysema. One animal killed forty hours after exposure showed less severe changes than the previous animals. The explanation of the rapid warming of inhaled cold air and of the occurrence of relatively mild and localized injury following the inhalation of cold air lies in the fact that dry air has an extremely low heat capacity and that the number of calories required to produce a great rise in the temperature of dry air can be provided by the heat derived from the cooling of a small amount of tissue by a few degrees. From these experiments it may be inferred (*a*) that it is unlikely that significant injury to the air passages of men would result from the breathing of air at any degree of coldness likely to be encountered in nonexperimental conditions so long as it was inhaled through the nose or between partially closed lips and (*b*) that, even though extremely cold air were inhaled rapidly through a widely opened mouth, it would be warmed to a point well above freezing by the time it reached the bronchi.—*Effects of Cold Air on the Air Passages and Lungs: An Experimental Investigation, A. R. Moritz & J. R. Weisiger, Arch. Int. Med., April, 1945, 75: 233.*—(*G. C. Leiner*)

Interstitial Emphysema and Subpleural Hematoma.—An 8 year old girl, who had been suffering from bronchial asthma for some years, had a severe asthmatic attack following which she complained of pain in her neck and chest; she developed dyspnea, swelling of the neck, a change of her voice. The diagnosis of subcutaneous, pulmonary and mediastinal emphysema was made. Five days later an X-ray examination of the chest was done which revealed a triangular paramediastinal shadow above the right diaphragm. The shadow had disappeared ten days later.

This shadow was believed to be produced by a subpleural hematoma. Two more cases of subcutaneous emphysema in bronchial asthma are described.—*Über interstitielles Emphysem und subpleurales Hämatom bei Asthma bronchiale. Ein Beitrag zur Differentialdiagnose bei paramediastinalem basalem "Dreieckschatten", L. Wallden, Upsala läkaref. förh., June 15, 1942, 47: 271.—(G. C. Leiner)*

Pulmonary Cysts. — Bronchopneumonectasis includes cysts of congenital (malformations) and acquired origin (deformations), classified as bronchiectasis, pneumonectasis and bronchiolopneumonectasis. The authors divide congenital cystic malformations into six groups: (*1*) pulmonary agenesis, (*2*) hypoplastic bronchiectasis (cystic anaplastic lung), (*3*) displastic bronchiectasis, (*4*) displastic bronchiolopneumonectasis (gaseous cysts), (*5*) hyperplastic bronchiolopneumonectasis (polycystic lung), and (*6*) displastic pneumonectasis (bullous emphysema). In general it is difficult and sometimes impossible to know whether the disease is congenital or acquired. This is likewise true of the clinical-radiological differentiation of some anatomical types. Bronchiectasis and bullous emphysema are predominantly of the acquired form and perhaps gaseous cysts also. Infection may be the causal factor or merely a complication revealing the presence of bronchopneumonectasis. Infection is of considerable importance in the development of obstruction and the subsequent apparition, insufflation and suppuration of the gaseous cysts. Cyst formations are frequently present as potential cavities with walls relaxed or in position and therefore invisible in the roentgenogram or tomogram. The insufflation of air or an accumulation of secretions distends the walls to form actual cavities. The rapid appearance and disappearance of a cavity as well as sudden modifications in its size are usual in bronchopneumonectasis. Complicating infection often renders apparent both clinically and roentgenographically a previously silent bronchopneumonectasis. The disappearance of a cystic shadow frequently occurs during the resolution of an intercurrent infection. The narrow communications between gaseous cysts or emphysematous bullae and the bronchial airway explain the frequent development of obstructive mechanisms whenever an inflammatory process happens to alter the physical state of the mucous secretions. The prevention of intracystic infection is based on the treatment of infection and bronchial obstruction. Sulfonamides and bronchial drainage are the two chief recourses. The authors report in detail 10 cases, in 2 of which pulmonary tuberculosis was a complication.—*El factor infección en las formaciones quísticas bronchopulmonares, R. Vaccarezza & J. Peroncini, An. Cáted. de pat. y clin. tuberc., December, 1943, 5: 199.—(R. Kegel)*

Concussion of Lung.—The frequent occurrence of gross pathological changes in the lungs of soldiers dying of wounds other than thoracic has stimulated this inquiry into the so-called "blast" lung. In a consecutive series of 87 autopsies, 30 instances of "blast" lung were found. All deaths were in soldiers who had serious head or abdominal wounds, and a few cases had severe burns. Although the method of wounding was unknown in 7 cases, it was felt that their lesions were identical with those who were known to have been exposed to blast. None of the cases had external evidence of damage to the chest, and none presented pulmonary hemorrhages. Some of the comatose cases did show fine râles at the lung bases, but in many cases it was felt that the obvious injuries should not have killed the patient. X-ray studies consistently showed less abnormality than was seen at autopsy. The picture was that of a scattered bronchopneumonia. It is difficult to diagnose the condition clinically, but it may be suspected in any patient who shows typical X-ray lesions of a patchy bronchopneumonia without physical signs of such, and whose other wounds are of such a nature that his condition should be better than it is. All variations in extent of the lesions were seen. When the involvement was mild, the lesions

were confined to the lower lateral portions of the lung. In the severe cases several or all lobes were involved. Grossly the hemorrhages vary from pinpoint to large confluent areas. Subpleural hemorrhage was seen ten times. Microscopically intraälveolar hemorrhages and congestion of the capillaries stood out. Bronchopneumonia was superimposed four times. Other organs showed similar hemorrhages in 10 cases.—*Pulmonary Concussion ("Blast") in Non Thoracic Battle Wounds, O. Savage, Lancet, April 7, 1945, 248: 424.*—(*H. Marcus*)

Air Embolism.—Pulmonary emboli may arise in the pulmonary veins in connection with practically every form of injury to these vessels. This type of embolism occurs relatively frequently in connection with artificial pneumothorax either through air being injected directly into a vein or by air in the pneumothorax cavity pouring into an injured vein. Since negative pressure often prevails in the pulmonary veins, air can be sucked into them, for example, on injury to a vein on the surface of the lung in pleural empyema or on puncture of or incision in the pulmonary parenchyma. On the basis of experiments on rabbits it was found that the average pressure in the left ventricle during inspiration was lower than the atmospheric pressure, regardless of the position of the animal. The experiments also indicated that the pressure was lower in the parts of the pulmonary veins above the level of the heart and that therefore air embolism was more likely to arise if the lesion was situated high up in relation to the heart. Judging by experimental and clinical experience, the site of the air emboli in the systemic circulation is highly dependent on the position of the body when the embolism develops. The dangerous symptoms are caused by emboli in the coronary circulation of the heart and in the cerebral vessels. In experiments on cadaver specimens, the risk of embolism in these vessels was found to be least in the supine position with the head elevated about 30 degrees. In this position, nearly all the air followed the blood-stream through the aorta and only very small amounts of air passed to the coronary vessels and the carotids. The air does not pass through the ordinary capillaries in the systemic circulation, but only through relatively wide arteriovenous anastomoses. Most of the air is absorbed from the precapillary vessels and thus does not reach over to the venous part of the circulation. This absorption occurs within an hour or so. Due to conglutination of corpuscles, the interruption of the circulation may continue even after the air has been absorbed. The symptom picture is generally dominated either by cardiac disorder or by signs of cerebral irritation or dysfunction. Quickly changing anemic or cyanotic patches on the skin and sharply outlined anemic areas on the tongue are sometimes of great help in the diagnosis. Air can be demonstrated by ophthalmoscopy soon after embolism has developed. Death occurs soon after the attack, a few hours later, or in occasional cases a day or so later. The patients who recover generally show no permanent ill effects. In order to reduce the risk of air embolism in irrigation of an empyemic cavity, provision must be made for the free escape of the air and water beside the tube or syringe used for irrigation. The patient should lie so that the cavity being irrigated is below the level of the heart. Punctures in the pulmonary parenchyma should also be made below the level of the heart. If blood is secured in the syringe on pneumonocentesis, it is dangerous to extract the needle before the patient has been placed so as to bring the site of the lesion below the heart. The use of a thermocautery instead of a knife to make the incision reduces the risk of air embolism. The thermocautery should be faintly glowing. Positive pressure respiration prevents air embolism from outside air, but does not prevent it from developing as a result of a bronchovenous communication produced, for example, by puncture through the pulmonary parenchyma. The treatment of air embolism should first be directed at preventing the formation of new emboli.

Artificial respiration can then be given, if indicated. Oxygen respiration and heart tonics probably have a favorable effect by facilitating the absorption of the air emboli. It is uncertain whether heart or vascular tonics or vessel-dilating preparations can hasten the passage of the air through the narrow vessels of the peripheral circulation. Nineteen cases of air embolism or suspected air embolism are described. All of them occurred in hospitals in Sweden during the years 1925 to 1938 in connection with the treatment of pleural empyema, with puncture of the lung for pulmonary abscess and with lobectomy or pneumonectomy. The rate of fatal air embolism in the treatment of pleural empyema in this material was at least 0.1 per cent and probably higher. The corresponding figure in puncture of the lung for pulmonary abscess was about 3 per cent. (Author's Summary.)—*Gas Embolism Originating in the Pulmonry Veins, V. Bahr, Upsala läkaref. förh., January 15, 1944, 49: 259.*—(*G. C. Leiner*)

Pulmonary Emboli.—Of 154 cases of death diagnosed as due to pulmonary emboli the diagnosis was confirmed by autopsy in 132 cases. In 22 no embolus was found, whereas in 90 in which emboli had not been suspected clinically, they were found on autopsy; 133 of these cases came from the Surgical Service and the correct diagnosis had been made in 102 cases (32 had not been recognized clinically and 15 could not be confirmed by autopsy). On the Medical Service the correct diagnosis was made in 30 cases, emboli were not diagnosed clinically in 52 and in 7 the diagnosis could not be confirmed on autopsy. Out of the 22 cases of false embolic syndromes the real cause of death was:

	SURGERY	MEDICINE	TOTAL
Number of emboli confirmed by autopsy	133	89	222
Correct clinical diagnosis	102 of 117 =87%	30 of 37 =81%	86%
Not recognized clinically	31 of 33 =23%	59 of 89 =66%	40%

Surgical cases:

Acute pulmonary edema	3
Pulmonary tuberculosis	1
Subacute bacterial endocarditis	1
Cardiac failure	1
Ruptured heart aneurysm	1
Bronchopneumonia	7
Undetermined	1
Total	15

Medical cases:

Mitral disease	2
Cardiac malformation	1
Bronchopneumonia	2
Undetermined	2
Total	7

Clinically the diagnosis of pulmonary embolus was missed in 149 cases and death was ascribed to uremia, myocarditis, cardiac failure, cachexia, etc. It appears that, although false embolic syndromes do occur, they are much rarer than would appear from the literature. On the other hand, the great number of cases clinically not diagnosed correctly indicates that a pulmonary embolus not always assumes the classical dramatic picture. An embolus was lodged in the main trunk of the pulmonary artery 104 times and 55 times emboli were found simultaneously in the left and the right branches.—*Quelques reflexions a propos d'une statistique de 222 cas d'embolies pulmonaires mortelles autopsiées a l'Institut d'Anatomie Pathologique de Strasbourg de 1926 à 1936, L. Géry, R. Fontaine & E. Blum, Presse méd., April, 1940, No. 35, 390.*—(*G. Simmons*)

Endobronchial Foreign Bodies.—In order to remove magnetizable foreign bodies from the stomach and tracheobronchial tree a magnet of cast alnico was used. Alnico is an alloy of aluminum, nickel, cobalt and iron.

The magnet is 3.5 cm. in length, 0.5 cm. in diameter and is attached to a Levine tube through which a metal stylet has been inserted. If necessary, an inflating mechanism can be attached to the other end of the Levine tube. The use of the magnet is illustrated by a report of the removal of a hairpin from the stomach of a baby.—*A New Magnet for Foreign Bodies in the Food and Air Passages, M. Equen, J. A. M. A., January 13, 1945, 127: 87.*—(*H. Abeles*)

Bronchogenic Carcinoma in Tuberculosis Hospital.—During a ten-year period, 12 cases of proved primary bronchogenic carcinoma were admitted to the Norfolk County Hospital for Tuberculosis. The most important conclusions to be drawn from these cases were that there are no certain diagnostic features which differentiate tuberculosis and other chest diseases from carcinoma. All symptoms and signs found in suppurative chest diseases or in tuberculosis may be present in malignant disease. The X-ray findings, infiltration, atelectasis and cavitation, are common to all diseases of the lungs. For this reason it is of prime importance to institute bronchoscopy just as soon as carcinoma has been considered in the differential diagnosis. It is unwise to await the result of sputum cultures and guinea pig inoculations, because the time interval between the suspicion of carcinoma and bronchoscopy should be days and not weeks. If bronchoscopy is inconclusive, further diagnostic measures are immediately indicated.—*The Differentiation of Bronchiogenic Carcinoma and Pulmonary Tuberculosis, N. R. Pillsbury & J. D. Wassersug, New England J. Med., March 8, 1945, 232: 276.*—(*H. Marcus*)

Tumor of the Alveoli.—The tumor of the alveolar cells, also called diffuse or multiple primary carcinoma, is extremely rare. Only 40 cases have been described. The *intra vitam* diagnosis generally is lobar pneumonia, miliary tuberculosis or tumor-metastasis. The case described by the author was diagnosed even during the autopsy as bilateral pneumonia. The patient had suffered from a slight cough for several months. During the last fifteen days of his life he had a very severe cough and high temperature. The diagnosis was bilateral pneumonia. The autopsy showed almost complete hepatization of both lungs. The color was a yellowish gray. The mediastinal lymph nodes were small and showed only some anthracosis. The bronchi contained abundant mucopurulent secretion. In the liver several foci of various size, but perfectly round, were found. These also were of a gray-yellowish color. Only this accidental finding gave rise to the suspicion that the process in the lung was malignant. Microscopic examination showed that in the affected regions the alveolar linings were invested by a layer of tumor cells. The tumor cells themselves were atypical and rarely showed mitosis. Their form was from cubic to high-prismatic. They were nonciliated and had a basophilic protoplasma. Nowhere would those cells invade the connective tissue. The nodules in the liver were of exactly the same structure. The neoplastic tissue extended over great parts of the lungs, but no primary tumor was found. The histological structure is definitely distinct from the more common bronchogenic carcinomata. Despite the wide-spread disease of the lungs the extrapulmonary metastases were insignificant, although in this case they allowed the author to make the correct diagnosis. The genesis of these tumors is disputed. No primary focus can be found and probably the disease has multiple primary centres. The majority of the authors accept the theory that the tumor starts from the alveolar epithelium. It has been proved in animal experiments that certain cancerogenic substances can produce atypical neoplastic changes in the alveolar epithelium. There is a certain resemblance to epizootic adenomatosis (jaagziekte), which causes very similar lesions in the lungs of cattle. Its cause is probably a filtrable virus.—*Sobre un caso de tumor de las celulas alveolares del pulmon, F. Wenger, Prensa méd. argent., January 5, 1945, 32: 44.*—(*W. Swienty*)

Carcinoma of Lung.—An analysis is presented of 181 consecutive cases of primary carcinoma of the lung in which operation has been performed over a period of eleven years. Seventy-one cases, or 39 per cent, have been operable, whereas 110 cases, or 61 per cent, were inoperable at the time of exploration. The distribution according to sex showed a six to one ratio in favor of the male. The age incidence did not differ to any great extent from the age incidence of malignant growths elsewhere in the body. The signs and symptoms were, in order of frequency, cough, hemoptysis, pain, loss of weight, hyperpnea, pneumonitis, fever, tightness in the chest. Not only the development of a cough but also changes in the character of a preëxisting cough are significant findings. The pain is usually described as a constant dull ache deep in the chest. The hyperpnea is paroxysmal in character. It may possibly be explained by a plug of mucus occluding a bronchus at the site of a tumor and causing obstructive emphysema. There are no characteristic signs and symptoms of primary carcinoma of the lung but their recurrent nature demands an early, thorough investigation of the respiratory tract. The roentgenogram of the chest is the most important method for the diagnosis of primary pulmonary carcinoma. Positive findings were obtained in all cases. They may be caused by the tumor itself or by secondary changes such as atelectasis, bronchiectasis, pneumonitis or abscess. Next to the roentgenogram, bronchoscopy is the most important examination. It revealed a bronchogenic carcinoma confirmed by biopsy in 61 per cent of the patients in this series. If the growth cannot be seen, fixation or deformity of the bronchial tree may give valuable information. There were no untoward effects due to the bronchoscopies. Bronchography may be useful to demonstrate occlusion of a bronchus which does not produce a shadow in the roentgenogram. It was not necessary to resort to bronchography in this series. Aspiration biopsy should not be used since it is generally uninformative and very dangerous. Exploratory thoracotomy was performed in 25 cases in which no definite diagnosis could be made by other means. If palpation or observation do not reveal the true nature of a lesion excision of the involved area should be carried out. There was no complication following this procedure. The pathological examination revealed two types of lesions. The majority of the tumors occurred at or adjacent to the hilum. They were of bronchogenic origin. They grew either intrabronchially into the lumen of the bronchus and toward the trachea arising from the bronchial mucosa or they grew along outside of and around the bronchus originating extrabronchially in the wall of a smaller bronchus and breaking through its wall. The minority of the tumors occurred in the periphery of the lung. They grew centrifugally apparently arising in the alveolar lining cells. The first symptoms produced by a primary carinoma of the lung depend upon the type of tumor. The peripheral tumors may remain asymptomatic until they invade the pleura, the chest wall or the brachial plexus. The hilar tumors of the intrabronchial type will cause respiratory symptoms, the ones of the extrabronchial type will interfere with the function of the mediastinal structures. In 70 per cent of the patients on whom pneumonectomy was performed there were metastases to the bronchial and tracheal lymph nodes which were removed together with the affected lung. In 110 cases that were found to be inoperable at the time of exploration, in addition to regional metastases there were metastases in other organs. Microscopically, 64 per cent of the tumors were classified as flat or squamous cell carcinoma and 36 per cent as adenocarcinoma; the latter group included the adenocarcinomata, oat cell types and cylindric cell carcinomata. The prognosis as to the length of life was better in the group of patients with a tumor of the flat or squamous cell type. The only efficacious treatment of primary carcinoma of the lung is pneumonectomy with removal of the regional lymph nodes. Medical therapy is only palliative, radiation

therapy is of no benefit. During a period of eleven years 71 pneumonectomies were performed for carcinoma of the lung. The postoperative mortality rate was 21 per cent. With the improvement of technique and the use of chemotherapy a decrease of the postoperative mortality rate can be expected.—*The Present Status of the Surgical Treatment of Primary Carcinoma of the Lung, W. F. Rienhoff, Jr., J. A. M. A., December 30, 1944, 126: 1123.*—(*H. Abeles*)

Primary Lung Cancer.—In the Mexico City General Hospital a special service for nontuberculous diseases of the chest has been founded. Systematic X-ray examinations, iodized oil studies and bronchoscopy are done in every patient between 40 and 60 years of age who presents symptoms of chronic nontuberculous bronchopulmonary disease. Occasionally, thoracocentesis and biopsy have been necessary to establish the diagnosis. Within three years 48 cases of pulmonary carcinoma were detected. The diagnosis has been confirmed either by biopsy or by autopsy. Among the 48 cases, 36 (75 per cent) were men and 12 (25 per cent) were women. The ages ranged from 33 to 68 years with an average of 53 years. The younger patients showed extensive and early metastasis which led rapidly to the fatal conclusion. The only symptom of one 33 year old patient was pain in the right arm produced by pressure on the brachial plexus by early metastasis in the supraclavicular ganglia. The primary neoplasm was found in the mucous membrane of the right upper bronchus. Death occurred after four months. Older patients showed a relative tolerance to the disease. A man of 64 years had signs and symptoms of bronchial carcinoma seven years prior to his admission. But thorough exploration of the mediastinum during operation did not reveal any metastasis. The localization of the primary neoplasm was in the right hemithorax in 30 cases (63 per cent), in the left chest in 17 cases (35 per cent) and in the trachea in one case. In 36 cases, the site of the tumor was established by bronchoscopy; 16 cases showed localization in the right lower bronchus, 18 in the right upper bronchus, 10 in the left lower bronchus, one in the left upper bronchus and one case in the trachea. Three cases of high localization and early supraclavicular metastasis were characterized by pain in the shoulder, Horner's syndrome, atrophy of the muscles of the hand and destructive processes in the ribs or in the neighboring vertebrae. Etiologically tobacco abuse is of importance, as it was found that all cases presented by the authors were heavy smokers. Bronchial carcinoma does generally not produce early metastasis. The time for surgical intervention (between the beginning of the disease and the appearance of distal lesions) is therefore long. Metastases takes place via lymphatics or blood-stream. The latter explains the frequency of cerebral metastasis in cancer as well as in pulmonary abscess. The localization of the metastases was generally in the mediastinum and supraclavicular lymph nodes, in the liver and in the brain. There was generalized carcinomatosis in one case. The majority of the cases had come to the authors after they had been treated for pulmonary tuberculosis for a long time. The main symptoms which made it possible to make the correct diagnosis were persistent cough with blood-tinged sputum or repeated hemoptysis. Only 30 per cent of the cases did not have blood in the sputum. Dyspnea and pain, slight afternoon temperature are almost constant symptoms and are explained by the presence of pulmonary suppuration, bronchiectasis or abscess cavities. The examination shows atelectasis or pulmonary consolidation with exudative bronchoalveolitis, destruction of parenchyma with suppuration, dilatation of the bronchi and pleural thickening. All operative and autopsy specimens showed that the tumor started in the bronchial mucosa, generally at the branching point of a primary or secondary bronchus. Bronchoscopy showed two main types of tumor: a polypous tumor which obstructed the bronchial lumen or an infiltrating tumor with irregular thickening of the bronchial wall and concentric stenosis. The

differential diagnosis has to exclude tuberculosis, syphilitic mediastinitis, scars from chest wounds, congential stenosis or benign tumor. A biopsy by lung puncture has been done four times and has given positive results in two: one sarcoma and one carcinoma. The prognosis is always fatal. Deep X-ray and radium treatment were of no benefit. Endoscopic resection is of no avail, as the endobronchial portion of the tumor is only small in comparison with the peribronchial process which cannot be removed. Pneumonectomy or lobectomy are the operations of choice. They should be done early. The increase of pulmonary cancer is due, in the authors' opinion, to improvement in diagnostic methods and not to factors depending upon modern civilization.—*Nuestra esperiencia en el cancer primitivo pulmonar, J. Gonzalez M. & A. Celis, Rev. mex. de tuberc., May–June, 1944, 6: 59.—(W. Swienty)*

Transthoracic Biopsy in Cancer of Lungs.—The symptoms of early cancer of the lungs are intense cough, bronchial catarrah with mucous hypersecretion, frequent attacks of asthma and dyspnea, repeated and sometimes abundant hemoptysis. Sibilant râles and rhonchi are generally present but may be absent if the tumor is situated in the periphery. The late complications and their symptoms are atelectasis, abscess, suppuration, pleural pain and exudate, compression of the mediastinal structures and distant metastasis. Bronchopulmonary carcinoma may be mistaken for tuberculosis, pleurisy, lung abscess or gangrene. In the absence of pathognomonic X-ray findings these errors are understandable. It is important to make the correct diagnosis early. Malignancy must always be considered if a patient of more than 40 years of age has symptoms of persistent bronchial irritation and pulmonary tuberculosis has been ruled out. For early diagnosis sputum examination, bronchography, bronchoscopy, planigraphy and bronchoscopic biopsy are necessary. Other diagnostic procedures of value are exploratory thoracotomy and aspiration biopsy. There are conflicting opinions about the advantages and dangers of the biopsy by transthoracic aspiration. The authors have not seen any untoward effects in their cases. In all operable cases thoracotomy should be done. Aspiration by biopsy is necessary where the bronchoscopy and the sputum examination give negative results. In the very early stages of bronchopulmonary malignancy, there are not enough diagnostic criteria to warrant an exploratory thoracotomy. In the late stages the tumor may be inoperable due either to its extent or due to metastases. In all these cases the authors use the transthoracic aspiration biopsy. Seven cases are presented. Six were more than 50 years of age and one 36 years. Six had blood tinged sputa, one hemoptysis from the beginning. All had pain in the affected hemithorax. One case simulated a pulmonary suppuration, one a pleurisy and 5 pulmonary tuberculosis. Of the latter, 2 were treated with pneumothorax, one with phrenemphraxis, one with gold salts and one with rest. In these cases no endobronchial lesion could be found. In 6 cases the allergy to tuberculin was low. In all 7 cases the aspiration biopsy proved the diagnosis. Six were epidermoid epitheliomata and one an adeno-carcinoma.—*Canceres pulmonares simulando diversas neumopatias, Su diagnostico por puncion transtoracica, A. Fernandez Conde & R. Meneses Mañas, Rev. cubana de tuberc., October–December, 1944, 8: 571.—(W. Swienty)*

Metastatic Cancer.—The roentgenological appearance of lymphatic spread of metastatic carcinoma is characterized by diffuse linear densities and fine miliary nodules involving mostly the lower and central portions of the lungs. In most cases the hilar nodes are invaded and enlarged. As a rule, both lungs are equally affected; sometimes, particularly if the primary tumor is in the lung, the lymphatic spread is limited to, or predominant on, one side. In the absence of known primary carcinoma, the roentgenological diagnosis may be difficult and a number of conditions (miliary tuberculosis, pulmonary edema and

congestion, sarcoidosis, pneumonoconiosis, bronchiectasis, etc.) have to be excluded before arriving at the diagnosis of lymphangitic carcinoma. In 70 per cent of the cases the primary tumor arises in the stomach. It is generally believed that the carcinoma cells reach the hilar nodes through the efferent, and not through the afferent, lymphatics. Of the two lymphatic systems of the lung, the deep (intrapulmonary) and the superficial (pleural), the former is predominantly involved. Macroscopically, the affected lymphatics appear as gray lines, arranged in a mosaic pattern; the bronchi and blood vessels are surrounded by a cuff of tumor. In microscopic sections the lymphatics are dilated and contain metastatic carcinoma cells. In some cases, wide-spread arterial involvement is present. With multiple tumor cell emboli, there is fibroblastic reaction in the adventitia and intimal thickening. The clinical symptoms of lymphangitic carcinosis (dyspnea, cyanosis, cough, cachexia) are frequently so severe as to overshadow those caused by the primary tumor. Occasionally right heart failure sets in. The clinical, roentgenological and pathological findings in 10 cases of lymphangitic pulmonary carcinosis are presented. It seems evident that the roentgenological manifestations are caused by the carcinomatous changes in the lymphatic system.—*Roentgenologic Appearance and Pathology of Intrapulmonary Lymphatic Spread of Metastatic Cancer, H. P. Mueller & R. C. Sniffen, Am. J. Roentgenol., February, 1945, 53: 109.*—(*P. Lowy*)

Hemosiderin Formation in Lung.—Three groups of rabbits were injected intratracheally with homologous blood, hypertonic dextrose solution and a mixture of the two. The lungs were examined after varying intervals. It was found that within four hours after the introduction of whole blood, hypertrophy, hyperplasia and desquamation of the alveolar lining cells occurred, reaching a peak in twenty-four hours and then receding. The red cells were disposed of rapidly, in part and perhaps entirely through the phagocytic activity of the activated alveolar lining cells, and there was no local hemosiderin formation. Hypertonic dextrose alone produced a transitory pulmonary edema and hyperemia for as long as twenty-four hours, with mild stimulation of the lining cells. A mixture of dextrose and blood produced a more pronounced lining cell activation and exudative response than that caused by either alone, and many intact erythrocytes were found in the alveoli after the second day. Hemosiderin was first observed after twenty-four hours as a faint blue discoloration (Gomori stain for hemosiderin) in the cytoplasm of the macrophages, both those free in the lumen and those attached to the septal wall. It gradually assumed a granular character and, by the end of a week, was seen as dark, compact blue masses. After the fourth day most of the intact red cells and free macrophages had disappeared. Those macrophages which remained attached to the septal walls appeared to undergo retrogression in size and were susceptible to reactivation as long as fourteen days after an episode of phagocytic activity. The hypertrophy and phagocytic activity of the alveolar lining cells apparently do not constitute an irreversible change so long as they retain their septal attachments.—*Formation of Hemosiderin in the Lungs: An Experimental Study, G. Strassmann, Arch. Path., August, 1944, 38: 76.*—(*D. G. Freiman*)

Thoracic Sarcoidosis.—The cutaneous lesions known as lupus pernio and the systemic disease known as sarcoidosis were first described as a clinico-pathological entity by Schaumann, who also called attention to the similarity between the X-ray appearance of chronic miliary tuberculosis and certain cases of sarcoidosis. It is now recognized that the histological unit of sarcoidosis is the "hard tubercle." It has been repeatedly shown that sarcoid tissue ultimately replaces lymphoid structures, and its predilection for the lymphoid tissue in the interlobar septa accounts for the diffuse streaking seen in the pulmonary roentgenogram. There may be extensive in-

filtrations and minimal or absent physical signs and symptoms. In some cases the radiographic shadows diminish or even completely disappear while in others they may persist without apparent change for many years. It is possible that a large number of cases of chronic or healed miliary pulmonary tuberculosis belong in this group. The average interval required for the lesions to disappear completely or almost completely is twenty-two months. After the clearing of the pulmonary infiltrations, recurrences are rare, but the appearance of new lesions, as hilar adenopathy is decreasing, is common. The heart is not infrequently involved, and there may be right heart failure due to diffuse pulmonary sarcoidosis. While the recognition of myocardial and pericardial lesions is often impossible during life, numerous cases of cardiac enlargement have been described which in the absence of murmurs or hypertension may represent myocardial sarcoidosis. Two cases in the authors' group developed cardiac enlargement while under observation. Twelve cases are presented, illustrative of the varied, but most frequent roentgenological appearance of thoracic sarcoidosis. Only cases confirmed by histological examination are included. For convenience they are grouped according to the roentgenological appearance when first seen. These groups demonstrate the chronicity and variable degrees of progression of the disease and also the definite tendency to complete healing; the hilar adenopathy and pulmonary infiltration of strand-like, reticulated, miliary, nodular or confluent type; the recession of hilar adenopathy with the extension of parenchymal infiltrations and vice versa. Involvement of the heart is suggested in 2 cases by the changes in size and shape of the cardiac shadow. Because of the roentgenological resemblance between sarcoidosis and other conditions, especially tuberculosis, diagnosis from X-ray studies alone is not justified. Histological findings should be the basis of diagnosis.—*Thoracic Manifestations of Sarcoidosis, S. S. Bernstein & M. L. Sussman, Radiology, January, 1945, 44: 37.—(G. F. Mitchell)*

Metabolism in Boecks' Sarcoidosis.—Sarcoidosis is considered a generalized, systemic disease. The skin lesions were the first to be described. Schaumann was the first to see all the manifestations as one disease, to realize that it may exist without the skin lesions, and that lymph nodes, tonsils, spleen, bone marrow and lungs are frequently involved. Recently pulmonary lesions, with or without lymph node enlargement, have been reported that have characteristics in common. Pinner reported similarities in many cases diagnosed as Boecks' sarcoidosis, uveoparotid fever, Mikulicz's syndrome, *osteitis tuberculosea multiplex cystoides* and a number of disseminated and lymph node lesions. The etiology of the condition remains unsettled. Pinner regards sarcoidosis as a form of "noncaseating tuberculosis." Pathologically the typical lesion is the so-called "hard tubercle" consisting microscopically of a collection of large, pale-staining, polygonal epithelioid cells which is usually sterile on culture and animal inoculation. The lesions may remain unchanged for long periods of time and heal by fibrosis and hyalinization. The signs and symptoms vary with the location and extent of the lesions, but include malaise, weakness, drowsiness, anorexia, weight loss, low grade fever, night sweats, dry mouth and generalized aches and pains. Laboratory studies may reveal a leucopenia with a relative increase in the monocytes or eosinophiles and with the red blood count below normal. Metabolic studies have revealed an elevation of the serum protein with a reversal of the albumin-globulin ratio. Harrell reported the blood calcium at or above the upper limits of normal in 6 of 11 cases and phosphatase levels above normal in 7. The prognosis is disputed, and the treatment is symptomatic. Three cases are reported by the writer in which metabolic studies were made. Two of the cases had uveoparotitis. Numerous laboratory and metabolic studies were made in the

hope of contributing toward the etiology of sarcoidosis. Guinea pig inoculations were negative. Antigen prepared from lymph nodes of one patient gave no positive intracutaneous reactions and no "anticutins" could be demonstrated. Only 8 proved cases of sarcoidosis of the heart have been reported, though there probably have been many others, and, while previous unrecognized rheumatic fever could not be definitely excluded, it is thought possible that the prolonged P-R interval of one patient might be an indication of sarcoid lesions in the heart of this patient. Leucopenia and eosinophilia were present in all the cases reported here. The red blood count and the hemoglobin were normal. The serum alkaline phosphates were elevated in the 3 cases, but the serum acid phosphates were not affected. The blood calcium, phosphorus, cholesterol, glucose and urea showed normal values.—*Sarcoidosis of Boeck, B. M. Stuart, Am. J. M. Sc., December, 1944, 208: 717.*—(*G. F. Mitchell*)

Pleuropulmonary Changes in Acute Nephritis.—In certain cases of diffuse glomerulonephritis, also called "war nephritis" or "trench nephritis," acute bronchopulmonary changes may present the main symptoms and completely overshadow the nephritis. The authors present 12 cases of diffuse, acute glomerulonephritis and one case of subacute hemorrhagic nephritis in which the pleuropulmonary complications were the foremost symptoms. These changes were pulmonary congestion in 8, cortico-pleuritis in 2, and broncho-alveolitis and bronchopneumonia in one case each. In all observations the renal and the pulmonary symptoms appeared almost simultaneously. The onset was always preceded by inflammation of the tonsils and the pharynx which, in the author's opinion, was the cause of the nephritis. The pathological changes of the lungs and the pleura presented no peculiarities and resolved in a very short time, except for one case (of hemorrhagic nephritis) who died. All symptoms were exclusively or predominantly in the base of the lungs. In the 2 cases of pleuritis the pleural fluid proved to be an exudate. This is of importance, since in nephritis with general edema frequently a transudate is found in one or both pleural cavities. The tonsillitis is the cause of both, the nephritis and the pleuropulmonary pathology. The pathogenic mechanism, however, is different; direct action of the germs in the lungs, and allergic response to bacterial toxins in the kidneys. The *Streptococus hemolyticus*, cause of the nephritis, is associated with pneumococcus, staphylococcus, *Micrococcus catarrhalis*, etc., which produce the pulmonary changes. The treatment is purely supporting. In 2 cases sulfanilamide was well tolerated and effective. The prognosis of the nephritis is not aggravated by the pleuropulmonary complications. — *Alteraciones pleuro-pulmonares en las nefritis agudas, R. Q. Pasqualini & M. C. Lascalea, Rev. Asoc. méd. argent., November 30, 1945, 58: 1074.*—(*W. Swienty*)

Pulmonary Changes in Malignant Hypertension.—The pulmonary findings in malignant hypertension are generally considered as due to passive pulmonary stasis caused by the changes of the heart. The authors believe that some of the changes in the lungs have to be interpreted as congestion or edema due to hyperazotemia. They describe 4 cases of malignant hypertension, in which the blood-urea was between 2 to 3 g. per cent. All had subcrepitant fine râles at the base or the middle portion of the lung, more frequently on the right side. The X-ray picture showed an irregular basal or central density, often bilateral, decreasing towards the periphery. This can lead one to believe that large nodules or inflammatory changes are present. The pulmonary changes in cardiac disease, especially left-sided heart failure, occupy the lower half of the pulmonary field, whereas the uremic lung always shows parahilar density. Here the X-ray findings may be the only means for a differential diagnosis. Differentiation from tuberculosis is evident

by the clinical and laboratory findings. Increase of the blood nitrogen level in these cases leads to an increase of the degree of pulmonary congestion, which responds promptly to a decrease of the blood nitrogen. In 8 cases of extrarenal hyperazotemia without hypertension the authors did not find any pulmonary changes.—*Aspectos radiologicos del pulmon en la hipertonia maligna, M. R. Castex & E. S. Mazzei, Prensa méd. argent., December 27, 1944, 31: 2637.*—(*W. Swienty*)

Pulmonary Changes in Retroperitoneal Tumor.—The author describes the case of a 30 year old woman who suffered from persistent elevation of temperature for seven years. An X-ray picture showed a left infraclavicular infiltrate and induration of the entire upper left lobe and marked accentuation of both hilar regions. This infiltration disappeared rather surprisingly from one X-ray examination to the other within a few days. Incidentally, a tumor of almost head-size was found in the left hypochondrium, moving with respiration. This tumor did not cause any symptoms. A pyelogram was taken and a renal tumor was diagnosed which compressed the stomach and the duodenum and shifted the entire upper portion of the intestine to the right, causing gastrointestinal disturbances. The patient suffered several sudden attacks of violent pain in the tumor region followed by cough, dyspnea and suffocation. A chest X-ray revealed an opacity covering the entire lower third of the left hemithorax and some haziness of the remainder of both lungs. Ten cc. of serous, yellow, clear transudate were aspirated. The patient recovered from this attack within four days. An X-ray film at this time showed complete reabsorption of the transudate, leaving only a slight veil-like haziness in the left lower chest. The fluid had completely disappeared but for a small amount in the left costophrenic angle. The author made the diagnosis of acute pulmonary edema due to "retroperitoneal paranephritic tumor." The possibility of acute dilatation of the left ventricle with exudation was also kept in mind. The patient then suffered another attack of dyspnea and cough, at which time an operation was done. A large tumor was found and a biopsy done. The tumor first gave the impression of a sarcoma and seemed to start from the capsule of the left kidney. Microscopic examinations showed that it was a pseudomucinous cystadenoma, possibily originating from Muller's duct. The patient died two weeks after the operation from a new attack of pulmonary edema. The pathological mechanism is explained by the diminished capacity of the veins in the abdomen due to the pressure and extent of the tumor and by the increased venous return. The author believes with Laubry and Doumer that the abdominal venous net acts as a reserve space and presents a security coefficient in case that the blood volume augments. In this case the blood volume did not increase, but the capacity of the security outlet diminished. The pulmonary capillaries were under a hypertension by the increased venous return. As they are separated from the atmospheric air by a thin membrane only, the alveoles are very easily filled with serosanguineous exudate. Also, it was evident from the microscopic examination that at the time of the crisis the tumor increased greatly in size due to massive interstitial hemorrhage. This in turn caused another decrease in the venous net of security.—*Comprobaciones quirurgicas del mecanismo patogenico de una imagen pulmonar fugaz, C. W. Gröbli, Prensa méd. argent., January 19, 1945, 32: 114.*—(*W. Swienty*)

Rupture of the Main Bronchus.—A case of rupture of a main bronchus without lesions of the thoracic cage is described. The patient survived the accident and the diagnosis was established by tomographic studies. Rupture of the main bronchi in the closed thorax is a rare occurrence. It is most frequent in young individuals, and the cause is mostly a crushing force (wheels of a vehicle), acting in antero-posterior direction. Fractures of ribs, of the clavicle, or of the sternum may be associated, but the external lesions may be insig-

nificant. A complete section of the bronchus occurs, with tear of the bronchial vessels and of the pleural covering of the hilum. In cases surviving the accident there is fibrous healing of the two stumps of the ruptured bronchus, retraction of the corresponding lung, overdistension of the contralateral lung and shift of the mediastinal organs. The most probable mechanism causing this lesion is a shortening of the antero-posterior diameter due to the crushing force and consecutive widening of the transverse diameter. The lungs closely follow the distending chest-wall until the bronchi tear. An increase in endobronchial pressure caused by spasm of the glottis may represent a contributing factor. When death does not follow immediately, the clinical picture is extremely grave and is characterized by dyspnea, cyanosis, shock, pain, hemoptysis, emphysema, pneumothorax and hemothorax. Treatment consists in relief of pain, deflation of hypertensive pneumothorax, repair of blood-loss. Sometimes exploratory thoracotomy or pneumonectomy is indicated.—*Ruptura de grandes bronquios en torax cerrado, O. A. Vaccarezza & A. A. Raimondi, An. Cáted. de pat. y clin. tuberc., 1943, 5: 76.*—(*L. Molnar*)

Lung Picture in Cardiac Insufficiency.—In cardiac insufficiency the X-ray picture of the lungs may simulate other pathological conditions. The author describes 2 cases in which the manifestations in the lung parenchyma and in the pleura responded to treatment of the underlying cardiac insufficiency. The first case was a woman of 65 years who complained of pain in the lower right chest, cough and occasional bloody sputum for the past six months. There was a soft, blowing murmur over the apex and a systolic murmur over the right base of the heart. Blood pressure was 160/100. Fluoroscopy showed a uniform opacity over the inferior third of the right hemithorax. The diaphragm was fixed. Within the right hilum a round, orange-sized shadow with blurred borders was seen. The electrocardiogram was normal. Differential diagnosis was pulmonary neoplasm, cardiac insufficiency or pulmonary infarct. After fifteen days of treatment with digitalis the lesions in the right lung field had almost completely disappeared. The digitalis treatment was considered as conclusive for the final diagnosis, which was cardiac insufficiency (despite the normal electrocardiogram). The second case was a 61 year old man who complained of dyspnea and of edema of the legs for one year. A soft systolic murmur was heard over all valves of the heart. There was extrasystolic arrhythmia. The blood pressure was 120/90, the pulse 98. There was a slight dulness over the base of the right lung and moist, crepitant râles over both lung bases, but especially over the right. The liver was markedly enlarged. The X-ray film showed a shadow within the region of the interlobar fissure on the right with perfectly clear borders and a small hydrothorax of the right base. This was diagnosed as interlobar pleurisy. The lesion disappeared on digitalis treatment. The author states that exudative lesions of the lungs or pleura may be simulated by cardiac insufficiency, that even with a normal electrocardiogram and a normal heart size cardiac disease should not always be excluded. In case of doubt, a fifteen-day treatment with digitalis is indicated.—*Imagenes pulmonares en la insuficiencia cardiaca, D. Fernandez Alfaro, Rev. cubana de tuberc., October–December, 1944, 8: 625.*—(*W. Swienty*)

Composition of Pleural Gas.—The authors determined the carbon dioxide-oxygen percentages in the pleural cavity of 76 patients with a therapeutic pneumothorax. In uncomplicated pneumothoraces the gas in the pleural space showed a CO_2 content ranging from 5.5 to 7.5 vol. per cent and an O_2 content of 2.0 to 4.5 vol. per cent. Following a pneumothorax refill stabilization occurred within forty-eight hours. In patients whose pneumothorax was complicated by a pleural effusion, the oxygen content of the pleural gas ranged from 0 to 1.15 vol. per cent. CO_2 values were fairly constant (9 to 12 vol. per

cent) in pure tuberculous effusions whether serous or purulent but varied from 7.5 to 18.7 vol. per cent in mixed infections. When a fistula without effusion was present the findings were characteristic: the CO_2 content was less than 5.5 vol. per cent and the O_2 more than 6.5 vol. per cent. An effusion complicating the fistula was always accompanied by a CO_2 content of more than 1 vol. per cent. A pneumonolysis had been done in 19 of the 27 patients with a fistula and pleural effusion. The diagnosis of fistula was confirmed in 5 of 8 patients who were examined postmortem.—*Gases pleurales y diagnóstico de la fistula pleuropulmonar, A. Soubrie & F. Labourt, An. Cáted. de pat. y clin. tuberc., December, 1943, 5: 277.*—(*R. Kegel*)

Gas Analysis in Bronchopleural Fistula.—The modern studies on diffusion and absorption of gases in body cavities are reviewed particularly the clinical application of gas analysis in the diagnosis in bronchopleural fistulae. The gas studies of the authors, in cases of pneumothorax with or without effusion, in presence or in absence of bronchopleural fistula, gave results identical to those obtained by Coryllos and Matsuzawa in similar circumstances. It seems noteworthy that bronchopleural fistula was found very often shortly after pneumonolysis. Repeated analysis of the pleural air is a reliable method in the diagnosis of bronchopleural fistula and deserves to be adopted as a routine investigation.—*La absorbcion de los gases en las cavidades del organismo: Diagnostico de la fistula pleuropulmonar, A. Soubrie & F. Labourt, An. Cáted. de pat. y clin. tuberc., 1943, 5: 120.*—(*L. Molnar*)

Respiration under Experimental Conditions.—The changes in vital capacity and maximum breathing capacity have been studied in 11 normal individuals submerged in water at different depths. Under these experimental conditions only the respiratory muscles are subjected to strain and consequently the results may be considered as a test of their action. The maximum breathing capacity has shown a much more accentuated progressive diminution than the corresponding values of the vital capacity. At depths over 30 cm. from the surface, parasternal pain was observed, probably at the site of the insertion of the pectoral muscles; 80 cm. below the surface respiration became impossible after one minute, because the expirations were larger than the inspirations. At depths ranging from 30 to 80 cm., in spite of quantitatively sufficient ventilation, dyspnea was noted. Similar results were obtained in experiments causing the subjects to perform expirations against resistance. This series of experiments shows that the reduction of the maximum breathing capacity is determined by muscular fatigue which causes diminution in the number of respirations. It is also demonstrated that the sensation of dyspnea is compatible with normal pulmonary ventilation and is caused, in these experiments and in normal activities reproducing similar conditions, by impulses originated in respiratory muscles subjected to excessive strain.—*La capacidad de ventilación pulmonar en condiciones experimentales desfavorables, F. Labourt & A. Lanari, An Cáted. de pat. y clin. tuberc., 1943, 5: 43.*—(*L. Molnar*)

Inspiratory Tonus in Anoxia.—Methods for plethysmographically recording the volume changes in the cat, and for measuring the chest circumference of the dog during procedures affecting respiration are described. During the reduction of O_2 content of inspired air from that of the atmosphere to about 8 per cent by rebreathing the volume of the cat's chest at the end of expiration increases by about 60 to 70 cc. This is three times the volume of a normal inspiration. During a similar diminution of O_2 in the inspired air of the dog a proportionate increase in expiratory circumference of the chest occurs. After vagotomy the anoxic increase in expiratory volume in the cat is about one-third as great as with the vagi intact. In the dog vagotomy totally eliminates the anoxic chest expansion. Crushing the nerves from the carotid chemoreceptors has little or no effect upon the

reaction. Hypercapnia appears to sensitize the animal to the low O_2 chest expansion reaction, but in the presence of a large excess of O_2 accumulated CO_2 does not produce an increase in the expiratory volume in cats or dogs. In some animals there is a small reduction in chest volume when the expirations become forceful. The human expiratory chest volume has been shown to enlarge during dyspnea. The suggestion that respiratory training to exhale more completely may be valuable to people at high altitudes is briefly discussed. (Author's Summary)—*Inspiratory Tonus in Anoxia, A. S. Harris, Am. J. Physiol., January, 1945, 143: 140.*—(*G. C. Leiner*)

Acute Abdomen Simulated by Pleuropulmonary Perforation.—A spontaneous pneumothorax caused by perforation of the visceral pleura may simulate under exceptional conditions an acute surgical abdomen. The symptoms are sudden abdominal pain which is severe and stabbing, vomiting, boardlike rigidity of the abdominal muscles, intestinal paresis, rapid pulse, profuse sweating and polypnea. If these symptoms are complicated by a high leucocytosis and marked shift to the left, it is understandable that a diagnostic error can be made and surgery performed. A small marginal pneumothorax may not even be detected in the X-ray film. The authors present 2 cases. In one the white count was 37,500 and went down to normal after aspiration of air. The tentative diagnosis had been perforated gastric ulcer until the X-ray film revealed a spontaneous pneumothorax. In the other case in which a laparotomy was performed the leucocyte count was 31,500 and rose to 68,500. The preoperative diagnoses had been: perforated gastric ulcer, perforation of the gallbladder, subphrenic abscess, liver abscess, acute appendicitis or acute pancreatitis. No abdominal abnormality was found. This case had been treated with a therapeutic pneumothorax and only after surgery a pleuropulmonary fistula with effusion was diagnosed. The mechanism of the abdominal pain in spontaneous or tension pneumothorax is still disputed. It may be caused reflexly by sudden compression of the mediastinum, by irritation of the sympathetic or pneumogastric nerves, by a viscero-motor reflex, by irritation of the inferior intercostal nerves which also innervate the proximal portion of the abdominal muscles, or by referred pain.—*Sindrome abdominal agudo por perforacion pleuropulmonar, A. F. Conde & E. A. Lastra, Rev. cubana de tuberc., July–September, 1944, 8: 504.*—(*W. Swienty*)

Spontaneous Pneumothorax.—Two cases of spontaneous pneumothorax due to metastatic sarcoma are reported. The first case was that of a 19 year old youth with a recurrent fibrosarcoma arising from the tendon sheath of the left *flexor pollicis longus*, for which the arm was amputated. Six months later the patient developed a chill and upper respiratory infection followed by dyspnea, right pleuritic pain and cough with bloody sputum. A roentgenogram of the chest revealed extensive metastases to both lungs and a right pneumothorax. Two weeks later, following some symptomatic improvement, he suddenly became markedly dyspneic and cyanotic, dying twenty-five minutes later. An autopsy revealed extensive metastatic fibrosarcoma of both lungs with bilateral pneumothorax. The second case was that of a 20 year old youth with an osteogenic sarcoma of the left femur for which a hip joint disarticulation was performed. A Welch bacillus infection followed, and the wound healed slowly. Seventeen months later a roentgenogram of the chest revealed metastatic foci, and two courses of roentgen therapy were administered. A right hydrothorax developed, accompanied by chest pain and cough. Several taps were performed revealing bloody fluid. Several weeks later he suddenly became dyspneic and was found to have a left pneumothorax. In spite of continuous suction drainage, he became rapidly worse and died. An autopsy revealed extensive metastatic osteogenic sarcoma of the lungs. The left lung was collapsed. Air injected into the trachea

escaped through a necrotic nodule in the left upper lobe. The rupture of a necrotic tumor nodule into a bronchus and into the pleural space, resulting in a small bronchopleural fistula, seems to be the most likely explanation for the occurrence of spontaneous pneumothorax in these cases. Such a complication is rare as evidenced by the lack of similar cases reported in the recent English and American literature.—*Pneumothorax Due to Metastatic Sarcoma: Report of Two Cases, T. F. Thornton & R. R. Bigelow, Arch. Path., May, 1944, 37: 334.*—(*D. G. Freiman*)

Pleurisy Caused by Brucellosis.—Physicians are more conscious of the wide spread of brucellosis since Gould and Huddleson have estimated that at least 10 per cent of the population of the U. S. A. have the disease and 1 per cent is clinically ill with it. From the various symptoms which can be caused by brucellosis the author has selected pleurisy. Patients affected with brucellotic pleurisy present cough and expectoration, pleural pain, subfebrile temperatures, loss of weight and secondary anemia. Four cases are discussed in which a pleurisy with effusion was present. The agglutination test was always positive in concentrations of 1:100 to 1:500. On treatment with specific vaccine the fluid completely disappeared in a relatively short time. The author considers it as very possible that some cases of brucellosis have been mistaken for tuberculous pleurisy, typhoid fever, rheumatic disease, nonpurulent pneumonia, syphilis or other infections. It is understood that tuberculosis has to be ruled out in every case, even if the agglutination test is found positive for Malta fever in low titres. The effusion can be either serofibrinous or hemorrhagic due to the tendency for hemorrhages which is one of the symptoms of brucellosis. It is suggested that this hemorrhagic diathesis is caused by a lack of ascorbic acid as it disappeared promptly with the administration of vitamin C. The prognosis is good. The fluid disappears without any sequelae. To ascertain the diagnosis of brucellosis the history of the patient and the existence of infected animals or other persons has to be searched for. The laboratory tests include the agglutination tests in the blood, in the cerebral-spinal fluid and in the effusion, the intracutaneous reaction and the blood culture from the pleural fluid and spleen. The agglutination tests should be regarded as positive even in as low a titre as 1 to 20 to 1 to 50. Finally, the author states that in each case of brucellosis a thorough study of the lungs and of the pleura by clinical and X-ray examination should be made routinely.—*Pleuresias brucelosicas, Contribucion a su estudio, I. Maldonado-Allende, Rev. Asoc. méd. argent., December 15, 1944, 58: 1107.*—(*W. Swienty*)

Hemothorax.—Although many theories have been advanced for the presence of clotting in some hemothoraces and its absence in others, no one theory will satisfy all questionable points. No matter what the pathogenesis of clotting may be, in the present series of 750 chest injuries, 52, or 70 per cent, had hemothoraces, and infection was present in 30 per cent of hemothoraces. Only 9 per cent of all hemothoraces were clotted. The incidence of clotting in hemothoraces rose sharply during 1944, when penicillin became more generally available. This led to the speculation whether penicillin, if used prophylactically without previous aspiration of blood, might be responsible for the clotting, although no such *in vitro* effect has been observed. Clotted hemothorax may be massive or loculated. The diagnosis may be suspected from the X-ray findings, but repeated unsuccessful aspirations usually prove its correctness. Although blood can be absorbed freely from the pleural cavity, and small, and sometimes even large hemothoraces may be resorbed, the usual course of events is the laying down of a thick layer of fibrous tissue on the parietal and visceral pleura. This prevents reëxpansion of the lung and is responsible for the "frozen chest" in which the lung loses most of its respiratory function. Evacuation of the clot with decortication of the lung has been found to be the most satisfactory treatment. Usually the entire lung must be de-

corticated, and only in small hemothoraces localized decortication will do. The operation is done through a thoracotomy with resection of part of the sixth posterior rib. The fibrous layer on the parietal pleura is two to three times as thick as on the visceral pleura and needs thorough removal as well. Suction drainage postoperatively, through two tubes, is of great importance. One tube is put into the upper chest, preferably into the second posterior intercostal space, to prevent residual pockets in the upper chest. A second tube is put low into the posterior chest at the conventional site. Negative pressures of 5 or 6 cm. of mercury are used. The results of suction drainage are far superior to underwater drainage in that lung expansion is more rapid and more complete. The identical treatment has also been applied to infected clotted hemothoraces and to closed and previously drained empyemata. It was found that decortication and suction drainage was a far more satisfactory treatment for these conditions, and expansion of the lung was the rule. In some cases expansion of the upper lobe only was obtained, and the empyemata became confined to small pockets which healed more promptly than a total empyema space would have. Closed empyemata are sterilized with penicillin before treatment, and infected hemothorax clots and open empyemata have been successfully treated with penicillin drip for twelve to twenty-four hours. The solution is administered through the two tubes later to be used for suction. Penicillin has been of great value in sterilizing pleural cavities infected with streptococci and staphylococci, but after decortication organisms are apt to recur. For this the penicillin drip is valuable, and the organisms tend to disappear as lung expansion proceeds. Coliform organisms, *B. proteus*, and *B. pyocyaneus* tend to disappear spontaneously after decortication and suction drainage, and *Clostridia* do not persist in adequately treated cases.—*Decortication in Clotted and Infected Hemothoraces, C. P. Thomas & W. P. Cleland, Lancet, March 17, 1945, 248: 327.*—(*H. Marcus*)

Traumatic Hemothorax.—When blood escapes into the pleural cavity rapid clotting takes place. Frequently, however, the clot remains liquid because the cardiac and respiratory motions defibrinate it and deposit fibrin on the parietal and visceral pleura. This is the case when bleeding occurs at a slow rate. When bleeding is rapid a solid clot forms which defies aspiration. Most traumatic hemothoraces yield to repeated early aspirations. If aspiration is not done early, or if a solid clot forms, surgical removal of the clot is indicated. This is done through a posterolateral incision, the exact location depends on the location of the clot. The sixth, seventh or eighth interspaces are most commonly used. Usually a well organized hematoma is encountered. Removal of this and its wall reveals a relatively normal pleura, and even at operation the lung will be seen to reëxpand partially, and the diaphragm will resume more nearly normal movements. Complete reëxpansion of the lung is effected with a short period of underwater drainage postoperatively. The results of such treatment are almost uniformly good permitting men to return to active duty within a short time. Untreated hemothorax results in a chronic respiratory disability. Low grade fever, persistent chest pain, anemia, dyspnea and asthenia are the rule. The decrease in respiratory function is striking. The affected hemithorax is partially immobilized, and the unaffected lung undergoes compensatory emphysema. Patients with untreated hemothorax cannot return to active duty.—*Traumatic Hemothorax, F. P. Coleman, Arch. Surg., January, 1945, 50: 14.*—(*H. Marcus*)

Hemothorax.—When blood is liberated into the pleural cavity by trauma, three immediate harmful effects take place: blood loss, pleural irritation and an expanding, space occupying lesion in the chest. Although no clotting is frequently observed in a hemothorax, deposition of fibrin on the lung, the parietal pleura and the diaphragm is usually prompt. The fibrin deposited on the visceral

pleura changes into fibrous tissue within a matter of days or weeks, and, unless prompt steps are taken to prevent this occurrence, the lung may become bound down by a rigid coat of fibrous tissue. The irritation which results from the presence of blood in the pleural davity, which here acts as a foreign body, causes the outpouring of a serous effusion. This augments the respiratory distress. Bleeding into the pleural cavity may come from the lung, from the chest wall or from the abdomen. Bleeding from the lung stops spontaneously and is rarely severe. Bleeding from the chest wall is often persistent, especially when it comes from an intercostal artery or the internal mammary artery. When the right leaf of the diaphragm has been penetrated by a missile and the foreign body has lodged in the liver, blood from the liver is apt to be pumped into the right pleural cavity with the respiratory movements of the diaphragm. Hemothorax is easy to diagnose if it is kept in mind that displacement of the mediastinum is a late sign that should not be awaited to make a diagnosis. Shock, pain, and dyspnea are early signs. The diagnosis is made by X-ray examination, but the patient must be in a sitting position. Lateral films are important. Even with these a hemothorax rarely gives the sharp straight outline which an empyema and other pleural effusions are apt to give. The margin of the hemothorax blends almost imperceptibly with the surrounding lung tissue, and a pulmonary hematoma may be mistakenly diagnosed. All hemothoraces should be treated promptly by aspiration, preferably within twenty-four hours. It is true that some hemothoraces resorb without treatment leaving no disability, but one cannot predict which ones are going to react this way. The only way in which poor late results can be avoided is by prompt immediate treatment of all cases. The majority of cases respond to repeated simple aspirations. As time goes on, aspirations become more difficult because of clots and fibrin, and a mistaken diagnosis of clotted hemothorax is often made. When hemothorax is very massive and bleeding continues under observation, it is best to evacuate blood, clots and fibrin through a thoracoscope. Air which enters the pleural cavity during this process should be aspirated. If bleeding continues slowly for many weeks, intercostal thoracotomy should be performed, the contents of the pleural cavity should be evacuated and the bleeding point secured, if possible. Even if this last procedure cannot be done, bleeding frequently stops following thoracotomy. Clotted hemothorax should be treated by thoracotomy and removal of the clot and decortication as soon as the diagnosis has been made. Retained foreign bodies should also be evacuated early, providing there are not too many and they are not located near the hilum.—*Hemothorax, N. R. Barret, Lancet, January 27, 1945, 248: 103.*—(*H. Marcus*)

Hemothorax.—When blood escapes into the pleural cavity it may remain liquid for a long time. This is due to the fact that, although clotting occurs, the clot is defibrinated through the mechanical action of the cardiac and respiratory movements. Fibrin is deposited on the visceral and parietal pleura, but the bulk of the contents of the cavity remains liquid. Early and regular aspirations reveal the fact that the hemoglobin content of the hemothorax is about 30 to 40 per cent less than the hemoglobin content of the blood. Repeated daily aspirations show that the hemothorax is quickly diluted by the outpouring of a serous effusion, and the hemoglobin content falls quickly. The fibrinogen content, on the other hand, rises progressively. The marked outpouring of fluid several days after wounding sometimes leads to the erroneous impression that bleeding is continuing. Aspiration quickly decides this question. A properly treated case should be dry after the fifth or sixth aspiration. If treatment has been delayed and fibrin has a chance to form, complete reëxpansion of the lung cannot take place. Evacuation of fibrin and clot through an intercostal incision must then be done, or in more severe cases thoracotomy and decortication of the lung must

be done to secure reëxpansion.—*Hemothorax, T. H. Sellors, Lancet, February 3, 1945, 248: 143.*—(*H. Marcus*)

Postoperative Empyema.—A study was undertaken to determine the value of penicillin prophylaxis in the prevention of empyema following transsection of the bronchi for lobectomy or pneumonectomy. Penicillin was administered intramuscularly every two hours daily for one week preoperatively and for two weeks postoperatively; the daily dose was 150,000 units of penicillin. No local application or instillation was used. Forty-eight patients had partial, single or multiple lobectomy or pneumonectomy. Seven patients died shortly after the operation. Forty-one patients were selected for analysis, 21 of them received penicillin prophylactically, 20 patients served as controls. Twenty-one patients had partial or total lung resection for bronchiectasis or multiple lung abscesses; 12 of them, 10 lobectomies and 2 pneumonectomies, were treated with penicillin. None of the 12 patients developed empyema. Nine patients served as controls in the series of suppurative pulmonary infections. All 9 patients developed empyema, 3 of them died. Fifteen patients had partial or total lung resection for tuberculous infection of the lung. Seven of the latter group were treated with penicillin, 2 of them developed tuberculous empyema. Eight patients served as controls, 3 of them developed pyogenic empyema. Five patients operated on for bronchogenic carcinoma had an uncomplicated postoperative course; 2 of them had undergone prophylaxis with penicillin and 3 patients had served as controls. Of the entire series of 20 control patients, 12 developed empyema. All 12 patients were treated with penicillin. Two received local therapy, they recovered promptly but developed fistulas later on. Three received systemic therapy, 2 of them died while one responded promptly. Seven received local and systemic therapy, 2 of them died, 3 showed slight improvement and 2 recovered promptly.—*Use of Penicillin in Prevention of Postoperative Empyema following Lung Resection: Report of a Controlled Study, W. L. White, W. E. Burnett, C. P. Baily, G. P. Rosemond, C. W. Norris, G. O. Favorite, E. H. Spaulding, A. Bondi, Jr. & R. H. Fowler, J. A. M. A., December 16, 1944, 126: 1016.*—(*H. Abeles*)

Mediastinal Emphysema.—Hamman called attention to 3 cases of interstitial emphysema simulating coronary artery disease; other investigators have mentioned the necessity of considering spontaneous mediastinal emphysema in the differential diagnosis of pain in the chest. The author has seen 8 cases in soldiers during the past three years. In 6, left-sided pneumothorax was demonstrated by X-ray. Four cases are reported, and the similarity of the condition to organic heart disease described. Macklin has described spontaneous mediastinal emphysema as occurring experimentally following overdistention of a lobe, the air entering the perivascular sheaths of the pulmonary vessels. The air bubbles gradually coalesce as they dissect their way and finally rupture into the mediastinum. This perivascular tunneling may also impede the circulation. Occasionally air may pass from the perivascular sheaths into adjoining connective tissue and thence to the pleura forming a subpleural bleb. In the mediastinum air tends to follow fascial planes, especially the sheaths of blood vessels. Thus, air may go into the neck, axilla and chest-wall and may also go between the parietal pleura and pericardium. Clinically, spontaneous mediastinal emphysema is characterized by the occurrence of sudden, often alarming precordial or substernal pain, the severity depending upon the degree of distention of the mediastinal tissues. It may radiate to the back, shoulders, neck or down the left arm and may last several hours or several days. Dyspnea, cyanosis and orthopnea may occur, but are not characteristic. The differential diagnosis depends entirely on the clinical examination. Air may be detected in the subcutaneous tissues of the neck and anterior chest-wall; the area of cardiac dulness may be replaced by hyperresonance,

but the pathognomonic sign is cracking or crepitant sounds heard over the precordium synchronous with the heart beat. Pneumothorax is present in some cases. If uncomplicated, there is no evidence of serious constitutional disturbances. The electrocardiogram reveals no significant change. X-ray films of the mediastinum are diagnostic. Diagnosis, however, may be difficult. This condition occurs chiefly in young people. There probably is a temporary functional coronary insufficiency. EKG changes were noted in 3 cases reported. No similar change is reported in the literature. Although not diagnostic, the electrocardiographic abnormality could be easily mistaken for serious myocardial damage. Similar changes have been reported in spontaneous pneumothorax apparently due to rotation of the cardiac axis.—*Spontaneous Mediastinal Emphysema with Pneumothorax Simulating Organic Heart Disease, H. Miller, Am. J. M. Sc., February, 1945, 209: 211.*—(*G. F. Mitchell*)

Pneumomediastinum. — Processes tending to produce overdistention of the lung may be associated with pneumomediastinum. Thus, in obstetrical labor, pertussis, obstructive tracheobronchitis, alveolar rupture may occur, with resulting escape of air along the vascular sheaths to the mediastinum. Local conditions, such as congenital deformities of the respiratory system, trauma, infectious processes, may play a part in the pathogenesis of pneumomediastinum. The consequences are varied. Rupture of mediastinal blebs may lead to unilateral or bilateral pneumothorax, or else may cause escape of air along the vessels and thus produce subcutaneous and retroperitoneal emphysema, pneumoperitoneum or even air embolism. Positive pressure in the mediastinum impedes the return flow of blood to the auricles, by compressing the pulmonary and systemic veins. Dyspnea and cyanosis ensue, with distended neck veins and falling blood pressure. The percussion over the heart becomes tympanitic, the sounds distant; there may be a crackling sound, synchronous with systole. In adults, precordial pain is a frequent complaint. Coexisting pneumothorax tends to alleviate the symptoms, by lowering the mediastinal tension. Roentgenologically, air may be seen outlining the margins of the superior mediastinum in the frontal view; in the lateral view which more often gives conclusive results, encapsulated air is present immediately behind the sternum. Treatment consists of oxygen and stimulants, or, in severe cases, aspiration of air. Three cases of pneumomediastinum in newborn infants are reported, all of whom received vigorous artificial respiration of some form. Whether the artificial resuscitation caused the pneumomediastinum, or the latter gave rise to the cyanosis necessitating artificial respiration, is difficult to determine.—*Pneumomediastinum in the Newborn, R. M. Lowman & C. S. Culotta, Am. J. Roentgenol., January, 1945, 53: 7.*—(*P. Lowy*)

Mediastinal Teratoma.—A case is reported of mediastinal teratoma in a 29 year old man admitted to the hospital with cough of three weeks' duration, aggravated by the prone position and worse at night, and swelling of the neck. A 10 by 6 cm. mass was present in the left anterior cervical triangle, displacing the trachea and larynx to the right and extending substernally. The neck veins were engorged and those of the left upper chest and arm dilated. A roentgenogram of the chest revealed a dense homogeneous mass in the upper mediastinum extending into the neck. Biopsy of a lymph node from the left side of the neck showed replacement by sheets and cords of undifferentiated cells with numerous mitoses. A course of roentgen ray therapy (3525 r over a period of seven weeks) produced a regression in size of only 2 cm. The patient died following an episode of dyspnea. An autopsy revealed a hard tumor mass measuring 18.5 x 17.5 x 10.6 cm. extending downward over the pericardium and upward into the neck, surrounding the ascending aorta and part of the arch, but not involving the thyroid or lung. The testis was negative. Microscopic examination of the tumor revealed a matrix of myxomatous and loose fibrous

tissue containing numerous cysts lined with epithelium ranging in type from stratified squamous to columnar and pseudostratified columnar. In other areas there were cells resembling adrenal cortical cells, endometrium and choriocarcinoma. The tumor appears to have arisen from all three germ layers.—*Mediastinal Teratoma, S. J. Wilson & R. Cares, Arch. Path., February, 1945, 39: 113.*—(*D. G. Freiman*)

Right Diaphragmatic Hernia.—In a 22 year old girl with the symptoms and signs of pulmonary and intestinal tuberculosis, the chest X-ray examination showed a picture similar to a right hydropneumothorax. The possibility of a pulmonary or pleural suppuration, or of a subdiaphragmatic abscess was considered. A later chest X-ray examination, one month before the patient's death, revealed two semi-circular opacities at the place of the previous "hydropneumothorax." The exact diagnosis was made after death only. The autopsy showed: Caseocavernous tuberculosis of the lungs with general amyloidosis; tuberculous ulcerations of the ileum; partial aplasia of the anterior part of the right diaphragm with herniation of the hepatic flexure of the colon. Right diaphragmatic hernia is found very rarely as compared to the left.—*Hernie diaphragmatique droite, L. Rousseau & M. Giroux, Laval méd., February, 1945, 10: 89.*—(*G. C. Leiner*)

Anomalous Pulmonary Veins.—Two instances of anomalous connection between the pulmonary and systemic venous systems, discovered at autopsy, are described. In one of these a vein draining most of the right upper lobe entered the right side of the superior vena cava; in the other most of the tissue of the upper lobe of the left lung was drained by a vein communicating with the left innominate vein. In both cases death was due to cardiac decompensation on the basis of rheumatic heart disease, and no symptoms that might have been attributable to the anomaly were recorded. By comparing the cross-sectional area of all pulmonary veins with that of the anomalous vein, the percentage of oxygenated blood shunted to the right side of the heart was estimated at 26.1 per cent in the first case and 20.1 per cent in the second. Seven cases encountered in the literature not included in a review by Brody (Brody, H.: Arch. Path., 1942, *33:* 221) are briefly summarized.—*Anomalous Pulmonary Veins, C. W. Hughes & P. C. Rumore, Arch. Path., June, 1944, 37: 364.*—(*D. G. Freiman*)

Vagotrigeminal Pain Reflex.—The irritation of the vagus in a region where its sensitive fibres are related exclusively to the functions of the vegetative life causes pain in the territory innervated by the trigeminus. These pains are caused, according to the author, by a vagotrigeminal pain reflex. Head studied the relationship between cephalic pain and the diseases of the thoracic and abdominal organs. Though Head advanced the hypothesis that in such cases the afferent stimulus might be conveyed by the vagus, a definite explanation was not given. The author says that there is a vagotrigeminal pain reflex that explains these cases of trigeminal neuritis. A patient with a bronchial fistula is presented in whom pain in the territory of the trigeminus appeared after mechanical or chemical irritation of the bronchial stump. Local anesthesia prevented the development of pain upon irritation. The author emphasizes the following points: In the patient presented the pain was unilateral and homolateral; no exophthalmos, midriasis, marked flushing of the face were noted; no pain was referred in other regions. All these facts indicate that the afferent pathway was not the sympathetic system. The proximity of the bulbar nuclei of these two nerves might explain the possibility of an afferent stimulus conveyed by the fibres of the region influencing the trigeminus.—*Sobre a existencia de um reflexo doloroso vago-trigeminal e de uma neralgia facial broncogenica, A. Amorim, Rev. brasil. de tuberc., May-June, 1944, 93: 121.*—(*P. B. Franca*)

Aortectomy.—The first case of a successful removal of an aneurysm of the thoracic aorta

is reported. A 19 year old student complained of fatigue following an episode of "flu." Physical examination revealed a loud blowing systolic murmur audible throughout the chest, best heard over the pulmonic valve area, and tachycardia. The blood pressure in the right arm was 160/72. It was not obtainable in the legs. Pulsations of the intercostal and internal mammary arteries were palpable. Pulsations of the arteries of the lower extremities were weak. The chest film showed an ovoid, nonpulsating mass, 6 by 8 cm, at the left border of the mediastinum at the level of the fifth, sixth and seventh vertebrae. The inferior borders of the lower ribs were eroded bilaterally. The Kahn test was negative. The patient was seen again two years later during which period he had led a normal life but had noted mild dyspnea and palpitation on exertion. At this time the chest film revealed an increase of the tumor to the dimensions of 10 by 8 cm. The preoperative diagnosis was aortic coarctation probably on the basis of a neurofibroma. Thoracotomy revealed a saccular aneurysm of the upper descending aorta, 11 cm. long and 8 cm. wide. Its attachment to the aorta was 7.5 cm. long. A smaller, daughter aneurysm arose from the large aneurysm. The aorta seemed constricted at its junction with the upper end of the aneurysm. Resection of the aneurysm and of the entire aneurysm bearing segment of the aorta was carried out. Reconstruction of the aorta was not done. During the postoperative period the blood pressure rose to 220/130. On the seventeenth postoperative day there was an episode of cardiac decompensation which responded well to treatment. About eight months after the operation the patient took up his regular activities. The chest film showed some symmetrical cardiac enlargement and an increase in the notching of the ribs. The blood pressure was 190/115. The urine was normal. Kidney function tests showed no abnormality.—*Aortectomy for Thoracic Aneurysm, J. Alexander & F. X. Byron, J. A. M. A., December 30, 1944, 126: 1139.*—(*H. Abeles*)

Mitral Stenosis and Silicosis.—The diagnosis of silicosis must be based on clinical evidence, not on roentgenological changes alone. In a 26 year old white man the roentgenological diagnosis of silicosis had been made. He had been working eighty-three days in a building in which a crusher of filter material was operated. The filter material consisted of finely ground silicate. The patient had far less exposure than 8 other persons who worked in the same building and of whom none developed signs or symptoms of silicosis. Physical examination of the patient revealed mitral stenosis. Fluoroscopy showed enlargement of the left auricle. Roentgenological examination revealed accentuation of the pulmonic conus and of the left auricular outline. There was diffuse mottling throughout both lungs, a picture similar to that found in silicosis. The pulmonary changes in this case were apparently due to congestion resulting from mitral stenosis.—*Pulmonary Roentgenographic Changes Due to Mitral Stenosis Simulating Those Due to Silicosis, H. W. Ryder & H. G. Reineke, Am. Heart J., March, 1945, 29: 327.*—(*G. C. Leiner*)

Subacute Pulmonary Endarteritis.—Clinical cases in which *Streptococcus viridans* has produced isolated vegetations on the intima of the pulmonary artery are infrequent. It is not unusual, however, to have a spread from the pulmonary semilunar valves for some distance up the pulmonary artery. Isolated vegetations situated in the pulmonary artery above the valves are associated with pathological lesions of a varied nature, and of them congenital defects in the heart and great vessels are the most important. A case of *S. viridans* endarteritis is reported in a 4½ year old white girl. This patient died approximately eight weeks after the onset of the disease. The postmortem showed subacute bacterial pulmonary endarteritis with multiple septic infarcts in the right lung. No changes were found in the heart and great vessels which might account for the atypical localization of the vegetations. The vegeta-

tions in the pulmonary artery consisted, to a large extent, of necrotic tissue arising from the vessel wall. This is in accordance with the observations that the vegetations in endocarditis are derived largely from the tissue of the valves. In the present case, the lesions developed in the lesser circulation, involved an apparently previously healthy vessel wall, and occurred in an atypical location where undue mechanical stress could not be postulated. *S. viridans* localized, therefore, in a manner resembling that of the aggressive organisms encountered in acute endocarditis. This assumption also seems supported by the fairly rapid course of the disease. However, the appearance of the lesion and other important clinical and pathological findings are those usually found with subacute bacterial endocarditis. (With 2 plates.)—*Subacute Bacterial (Streptococcus Viridans) Pulmonary Endarteritis, A. E. Rhoden, Am. J. Path., May, 1945, 21: 507.*—(*J. S. Woolley*)

Exudative Pericarditis.—A new roentgenological sign of exudative pericarditis is described. It consists of a change of the heart shadow with respiration: The orthodiagraphic diameter of the heart decreases with inspiration and increases with expiration. A moderately large amount of pericardial effusion and a good respiratory motility of the diaphragm are necessary to produce this sign. It was seen in a 25 year old female in whom the clinical diagnosis of septicemia with pericarditis was made. The orthodiagraphic diameter of the heart was 15 cm. during the expirium and 12.5 cm. during the inspirium. At autopsy 300 cc. of fluid were found in the pericardium.—*Über Röntgensymptome bei exsudativer Perikarditis, L. Walldén, Upsala läkaref. förh., September 20, 1942, 48: 133.*—(*G. C. Leiner*)

Hemorrhagic Tuberculous Pericarditis.—Two cases of hemorrhagic pericarditis are described. Inoculation of the exudate into a guinea pig proved the diagnosis of tuberculosis. Both cases recovered, and the recovery is ascribed to the small number of the tubercle bacilli present and their mild virulence.—*Péricardites hémorrhagiques tuberculeuses curables, R. Dupérié, A. Fontan & R. de Lachaud, J. méd. Bordeaux, 1942, 119: 49.*—(*G. Simmons*)

Silicosis of Pericardium.—Silicosis of pericardium is rare. A 51-year-old white man, stonecutter, in whom a left pneumothorax had been induced for silicotuberculosis, was admitted to the hospital. On admission the chest roentgenogram showed a left hydropneumothorax with almost complete collapse of the lung, and diffuse nodular infiltrations throughout the right lung. Electrocardiographic examination revealed an inversion of the T wave and slurring of the QRS complex in lead II. The patient died with the signs of pulmonary insufficiency. On autopsy, silicotic foci and tuberculous cavities were found in both lungs. Silicotic foci were seen in the tracheobronchial and peribronchial lymph nodes. The heart weighed 360 g., the right ventricular wall measured 8 mm. in diameter, the left, 11 mm. The pericardial cavity was obliterated and the parietal pericardium was adherent to the left pleura. Microscopic examination showed silicotic foci within the visceral pericardium over the left ventricle. No silicotic foci were found in the myocardium. It is believed that these findings prove the communication between pleural and pericardial lymph vessels.—*Silicosis of the Pericardium: Case Report, Marguerite G. Stemmermann, Am. Heart J., May, 1945, 29: 642.*—(*G. C. Leiner*)

Erythema Nodosum.—The old view was that erythema nodosum is frequently, if not generally, an expression of rheumatism even when no other definitely rheumatic symptoms are present. More recently great doubt has been expressed regarding such specific association of the two conditions. The recognition of small epidemics of this condition led some observers to consider it a specific infectious disease. Isolation of a specific

causal organism, however, has never been confirmed. Studies revealed the fact that it is not infrequent for the eruption to be followed some months later by evidence of tuberculosis such as a pleural effusion or tuberculous meningitis. It has been shown in Scandinavia that the vast majority of children with erythema nodosum have strongly positive tuberculin reactions; in addition many cases present radiological evidence of enlargement of the hilar lymph nodes and, in a smaller number, shadows of primary tuberculosis. Several investigations have clearly shown that the occasional epidemics seen in schools and institutions are in the vast majority due to recent simultaneous infection with the tubercle bacillus. Very few of the attempts to demonstrate tubercle bacilli in the actual nodes have been successful and these few successes are probably explained by the assumption that the cultures were made at a time when there was a tuberculous bacillemia. As a result of these investigations the theory was put forward that erythema nodosum occurs during primary tuberculous infection at the time the patient first develops hypersensitivity to tuberculin. Punch (1941) observed that in a number of student nurses undergoing regular tuberculin-testing the change from a negative to a positive tuberculin reaction was marked by the development of erythema nodosum. However, in a series of 800 cases 5 per cent showed a negative tuberculin reaction. Furthermore cases of eruption have occurred in patients known to have been infected with tubercle years before and who show evidence of this old infection in the form of calcified hilar nodes. The occasional development of erythema nodosum after hemolytic streptococcal infections such as scarlet fever has been observed. Further, about 5 per cent of patients with coccidioidomycosis show typical erythema nodosum. Erythema nodosum is occasionally met with as an episode in the prolonged clinical histories of some cases of sarcoidosis. Occasionally, after the use of sulphonamides, an eruption indistinguishable clinically and histologically from erythema nodosum results—the eruption, however, fades away rapidly when the drug is stopped. In a series of 112 cases studied by the author, 38 were males and 74 females. Of these, 61 were strongly positive to 0.01 mg. of tuberculin. In 21 of the 61 the X-ray films showed enlarged hilar shadows and some of these also showed a shadow of the primary lesion. One patient had typical tuberculous cervical adenitis; one patient died later of tuberculous peritonitis and 4 cases subsequently developed a pleural effusion. Only 10 of the 112 cases had a history of acute rheumatism—in only 2 of these was there any evidence of a rheumatic relapse. The author concludes that erythema nodosum must be regarded as the result of a nonspecific reaction to a variety of infections or toxic agents and that it is not a specific disease.—*Aetiology of Erythema Nodosum, C. B. Perry, Brit. M. J., December 30, 1944, 2: 848.*—(*D. H. Cohen*)

Hodgkin's Disease.—A 44 year old, white man noted a lump on the midposterior aspect of the right calf in March, 1939. In August, 1939 it ulcerated and had a purulent discharge. Soon afterwards several more lumps developed in the surrounding region as well as on the opposite calf. A biopsy of the skin lesion was reported as idiopathic multiple hemorrhagic sarcoma. The patient received radiation therapy. In September, 1940 he first experienced chills, fever and sweating. He was admitted to a hospital in November, 1940. Physical examination revealed slightly enlarged submaxillary lymph nodes. The skin of the left buttock and of the right popliteal space showed multiple shallow ulcers. Many brown and grayish-black spots were present on both legs. There was a moderate secondary anemia. A blood culture was negative. A roentgenogram of the chest was essentially negative. Three days after admission the patient developed erythematous skin lesions which became nodular in character within twenty-four hours. Microscopic examination of the new lesions revealed a slight endothelial and fibroblastic proliferation about small blood vessels. The older lesions showed

findings typical of Hodgkin's disease. On the eighth hospital day hundreds of small papular nodular erythematous lesions appeared. Chills and fever were associated with the new eruption. The patient died eight days later. The autopsy diagnosis was Hodgkin's disease of the skin with metastases to the abdominal and pelvic lymph nodes, trachea, lungs, pleura, esophagus, stomach, liver, spleen and lumbar vertebrae. The occurrence of terminal blood-stream spread without any evidence of embolic cellular element dissemination is consistent with a virus etiology of Hodgkin's disease.—*An Unusual Case of Cutaneous Hodgkin's Disease with Terminal Blood Stream Spread, S. R. Bersack, J. A. M. A., December 16, 1944, 126: 1025.*—(*H. Abeles*)

Sarcoidosis.—Theories regarding etiology are reviewed and summarized in four categories: (*1*) nonvirulent tubercle bacillus, (*2*) a nonspecific tissue response to various organisms, (*3*) disease of the reticulo-endothelial system, similar to Hodgkin's disease, and (*4*) a chronic infectious granuloma possibly due to a virus. The pathological lesion resembles a miliary tubercle, being similar to the lesions Sabin has been able to produce in guinea pigs by injection of the phosphatide fraction of the tubercle bacillus. In all cases in which caseation was present, tubercle bacilli could be demonstrated, but none were demonstrable in the noncaseating lesion. The author does not rule out sarcoid because of the presence of a caseating lesion. The great majority of cases are hyposensitive or anergic to tuberculin. Clinical features include insidious onset and extreme chronicity. The manifestations depend on the structures involved, most commonly lymph nodes, lungs, skin, or bones. The disease is not incompatible with longevity. A case is presented which suggests kidney involvement because of nocturia several times nightly over a period of several years, pyuria, cylindruria and hypertension. Dyspnea led to X-ray examination which revealed bilateral hilar node involvement with bilateral productive infiltrations. Inguinal lymph node biopsy confirmed the diagnosis of Boeck's sarcoid. The patient is alive and able to carry on moderate work two years after diagnosis and three years following the original symptoms.—*Boeck's Sarcoid, W. L. Meyer, Dis. of Chest, November–December, 1944, 10: 509.*—(*K. R. Boucot*)